A Study of

ACUTE INTESTINAL OBSTRUCTION

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CERTIFICATE

This is to certify that this dissertation in **"ACUTE INTESTINAL OBSTRUCTION"** is a work done by **DR. RASHEEDHA BEGUM. U.,** under my guidance during the period 2005-2007. This has been submitted in partial fulfillment of the award of M.S. Degree in General Surgery (Branch – I) by the Tamilnadu Dr. M.G.R. Medical University, Chennai – 32.

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CONTENTS

INTRODUCTION

Acute intestinal obstruction is an enigma with its versatile presentation and wide spectrum of management options available. It presents a constant challenge to the surgeon both in diagnosis and in judgment as regards the line of management for that specific patient, for those specific parameters and for that specific moment in the course of the disease. Intestinal obstruction is a dynamic condition where the parameters are changing as the clock ticks on. 'Time' means everything in intestinal obstruction and a delay in diagnosis or management increases the mortality. With a wide range of conditions known to cause obstruction, the surgeon should be aware of the commonest causes in the geographical region of presentation.

Keeping with these principles, this study hopes to discuss the various causes and clinical presentations, sift out the reliable signs and symptoms and the most common causes. Diagnostic modalities have reached new heights in present times, but our study focuses on using the available basic modalities to further the diagnosis. Different treatment modalities and their outcome in terms of death/morbidity have been tabulated.

AIM OF STUDY

1. To identify the cause of acute intestinal obstruction

2. To discuss the various clinical presentation

3. To discuss the various diagnostic modalities

4. Various modalities of management of acute intestinal obstruction

5. To study the outcome of patients with acute intestinal obstruction

REVIEW OF LITERATURE

HISTORICAL HIGHLIGHTS AND PIONEERS IN TREATMENT

1. HIPPOCRATES and CELSUS: (500 BC)

Hippocrates the father of medicine, and Celsus the most distinguished medical authors followed the pattern of egyption treatment which was the administration of purgatives and enemata for consecutive days once in a month to clear the bowels and as a prophylactive measure.

2. AMBROISE PARE (1510-1590)

A French physician who identified bowel obstruction for the first time and had reported a patient who died of twisted bowel.

3. HEISTER (172)

Described a successful resection of strangulated intestine with diversion of faecal stream.

4. PILLARE (1776)

He successfully made a caecostomy for a case of carcinoma rectum.

5. PAUL AND BLACK (1846 & 1892)

Proved that the exteriorisation of the colon is preparable to a major surgical procedure like primary anastomosis.

6. PAUL AND MICKULICKZ (1908)

Advocated the procedure of exteriorisation.

7. HENRI HARTMANN (1921)

He described the Hartmann procedure. It is an alternative procedure for recto sigmoid growth; it involves resection of the sigmoid colon with proximal diversion and over sewing of the distal stump.

8. MUIR (1968)

On table colonic lavage can be used to prepare the bowel intra operatively for a primary anastomosis. This technique was first described by him.

SURGICAL ANATOMY

The intestine which is the longest part of the digestive tube, is divided into the longer less distensible small intestine and shorter more distensible large intestine.

Small intestine

The small intestine extends from pylorus to the Ileocaecal junction. It is about 6 meters long.

The upper fixed part is the duodenum. The lower mobile part of the intestine is the Jejunum and Ileum.

Duodenum:

Duodenum is 25 cm long. It is divided into four parts:

1. First or superior part – 5 cms
2. Second or descending part – 7.5 cms
3. Third or horizontal part – 10 cms
4. Fourth or ascending part – 2.5 cms.

The Duodenum is mostly retroperitoneal.

Jejunum and Ileum

The Jejunum and Ileum are suspended from the posterior abdominal wall by the mesentry and therefore enjoy considerable mobility.

The Jejunum begins at the duodenojejunal flexure. The Ileum terminates at the Ileo caecal junction.

Large Intestine

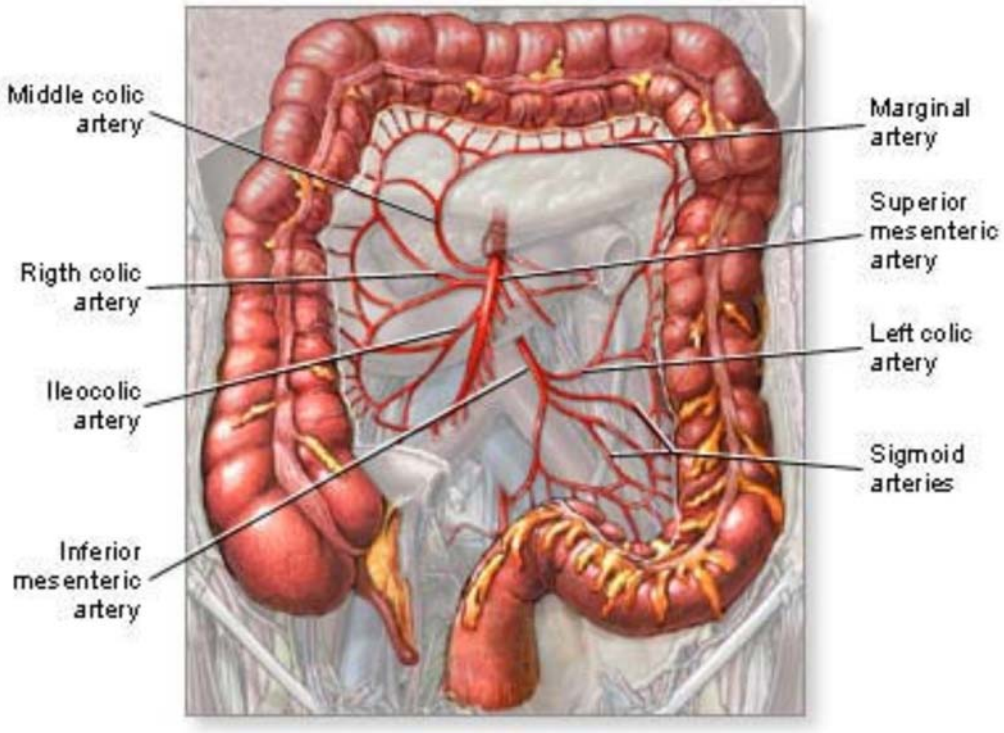
The large intestine extends from the Ileocaecal junction to the anus. It is about 135 cms – 150 cms long. (8)

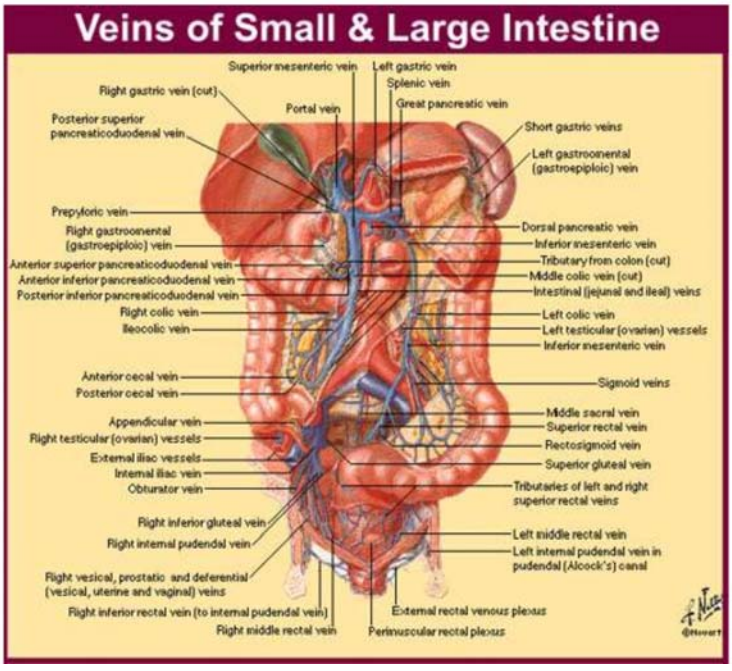
It is divided into

1. Caecum	-	6 cms
2. Ascending colon	-	12.5 cms
3. Transverse colon	-	50 cms
4. Descending colon	-	25 cms
5. Sigmoid solon	-	37.5 cms
6. Rectum	-	12 cms
7. Anal canal	-	4 cms

In the angle between caecum and the terminal part of ileum there is a narrow diverticulum called vermiform appendix.

LARGE INTESTINE BLOOD SUPPLY





The greater part of large intestine is fixed except for the appendix, transverse colon and sigmoid colon.

ARTERIAL SUPPLY

Duodenum – Up to the level of the Bile duct opening the duodenum is supplied by the superior pancreatico duodenal artery and below it by the inferior pancreatico duodenal artery.

Ileum & Jejunum - Supplied by superior mesentric artery.

- The caecum, Ascending colon, right two thirds of the transverse colon are supplied by superior mesentric artery
- The Left one third of transverse colon, descending colon upto upper one third of rectum are supplied by the inferior mesenteric artery.
- The middle rectal artery from anterior division of internal Iliac artery supplies the lower part of the rectum.
- The part of the anal canal above the pectinate line is supplied by the superior rectal artery.
- The part below the pectinate line is supplied by inferior rectal artery.

VENOUS DRAINAGE

The venous drainage of duodenum is into the splenic, superior mesenteric and portal veins.

The Jejunum and ileum are drained by the superior mesenteric vein.

The caecum, Ascending colon, right two third of transverse colon are drained by the superior mesenteric vein.

The left one third of transverse colon, descending colon, sigmoid colon, and upper one third of rectum are drained by the inferior mesenteric vein.

The lower two third of the rectum is drained by the internal Iliac vein.

The Anal canal is drained by the superior and inferior rectal vein.

LYMPHATIC DRAINAGE OF SMALL INTESTINE

Most of the Lymph vessels from the duodenum end in the pancreatico duodenal nodes. From here the Lymph passes partly to the hepatic nodes, and then to the coeliac nodes and partly to superior mesenteric nodes.

Lymph from Ileum and jejunum drains into numerous lymph nodes present in the mesentry, and along the superior mesenteric artery.

LYMPHATIC DRAINAGE OF LARGE INTESTINE

The nodes are arranged on a plan common to all parts of the large and small intestine.

They are numerous and arranged in three groups.

(a) proximal

(b) intermediate

(c) distal

Proximal nodes

These nodes are situated on the main blood vessels to the gut, (i.e) superior mesenteric, Ileocolic, right colic, left colic, middle colic, inferior mesenteric, superior rectal, sigmoid.

The Intermediate Nodes

The intermediate nodes are situated along the larger branches of the above named vessels.

The Distal Nodes

The distal nodes are situated near the gut between the numerous small vessels entering the gut.

ACUTE INTESTINAL OBSTRUCTION

DEFINITION

Impairment to the aboral passage of intestinal contents may result from either a mechanical obstruction or the failure of normal intestinal motility in the absence of an obstructing lesion (Ileus).

TYPES OF PRESENTATION:

A. Depending on onset:

   Acute

   Acute on Chronic

   Sub acute

   Chronic

B. According to the degree of obstruction

   Partial

   Complete

C. According to the absence or presence of Intestinal ischemia

Simple

Strangulated

D. Depending on Site of obstruction:

Small bowel

Colonic

E. According to the Level of obstruction:

High small bowel obstruction

Low small bowel obstruction

Large bowel obstruction.

F. Depending on blockade at both ends of an intestinal loop

Open loop obstruction

Closed loop obstruction.

G. Depending on the cause

- Dynamic
- Adynamic

Peristalsis may be absent-eg: paralytic Ileus

Non Prospulsive form of obstruction –

eg: mesenteric vascular occlusion (1)

AETIOLOGY:

Mechanical small bowel obstruction is responsible for most cases that need surgical intervention. The causes of bowel obstruction can be conveniently divided into intrinsic, extrinsic and intraluminal lesions.

CAUSES OF SMALL BOWEL OBSTRUCTION

1. Intrinsic Bowel lesions:

A. Congenital

1. Atresia
2. Stenosis
3. Malrotation
4. Meckel's diverticulum

B. Inflammatory:

1. Infections – e.g: Tuberculosis

2. Crohn’s disease

3. Ischemia

4. Radiation Injury

C. Traumatic:

1. Hematoma

2. Ischemic stricture

D. Neoplastic:

1. Primary Neoplasms

2. Metastatic Neoplasms

3. Peutz Jeghers syndrome

E. Miscellaneous:

1. Intussusception

2. Extrinsic Bowel Lesions:

A. Congenital bands

B. Adhesions

C. Hernias

D. Volvulus

E. Carcinomatosis

F. Abscess

3. Intraluminal:

A. Gallstone ileus

B. Enterolith

C. Bezoar

D. Swallowed foreign body

E. Balloons of intestinal tubes

F. Parasites e.g: Ascaris

CAUSES OF MECHANICAL LARGE BOWEL OBSTRUCTION

I. Within the lumen:

Faecal Impaction

Inspissated barium

Gall stone

Foreign body

II. Within the wall:

i. Tumours especially carcinoma

ii. Inflammation

i. Diverticulitis

ii. Crohn’s disease

iii. Tuberculosis

iii. Congenital causes

i. Adult Hirschsprung’s disease

iv. Ischaemia

v. Radiation

vi. Miscellaneous - Intussusception

III. Outside the wall:

Bands and adhesions

External hernias

Internal hernias

Volvulus

Tumours in adjacent organs or lymph nodes.

Most common causes of small bowel obstruction are hernias, post operative intra abdominal adhesions and neoplasm.

1. HERNIAS:

Inguinal, Femoral and ventral hernias and less frequently internal hernias are one of the common causes of Acute Intestinal obstruction in our country.

Relative Frequency of Specific types of Hernias Associated with small Intestinal obstruction in 4 series.

Types	**STEWARDSON N= 57**	**MUCHA N= 47**	**MCENTEE N= 59**	**GREENE N= 106**
Inguinal	54%	26%	46%	54%
Femoral	14%	9%	37%	24%
Incisional	14%	21%	10%	7%
Umbilical	16%	8%	3%	9%
Internal	---	34%	---	4%

OBSTRUCTED INGUINAL HERNIA

OBSTRUCTED FEMORAL HERNIA

Mucha reported that strangulated obstruction occurred in one third of the cases due to hernias whereas only 8% of patients with adhesive obstruction had strangulated bowel.

Brolin and colleagues found that each of the 22 cases of intestinal obstruction due to hernias was complete, whereas only 38% of patients with obstruction due to intra abdominal adhesions was complete. (23)

The obliquity of the inguinal canal and the higher frequency of occurrence makes inguinal hernia the most frequent variety associated with small bowel obstruction. The rigid fascial defect through which the herniated intestine must protrude makes hernias a particularly common cause of strangulation.

The occurrence of Small bowel obstruction in a patient without prior laparotomy should suggest a hernia as the cause. If no hernia is discovered on physical examination, internal hernias such as paraduodenal and obturator hernias must also be considered.

Retro anastomotic and parastomal hernias are also important causes of intestinal obstruction in patients who have had operative procedures in which mesenteric defects may be present.

OBSTRUCTED INCISIONAL HERNIA

SIGMOID VOLVULUS

2. ADHESIONS

The most common etiology of small bowel obstruction is intra abdominal adhesions following laparotomy, accounting for about 66% to 75% of cases. (23)

Causes of intra peritoneal adhesions:

1. Ischemic areas
2. Infections
3. Presence of Foreign bodies
4. Inflammatory disease
5. Radiation enteritis

Lower abdominal or pelvic procedures have a higher risk of post operative adhesive obstruction than do upper abdominal procedures.

3. NEOPLASMS:

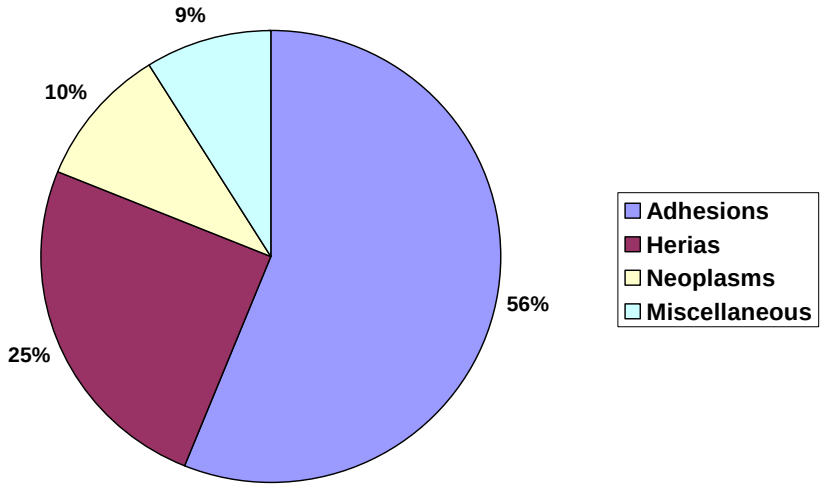
In contrast to colonic obstruction, neoplasms are a relatively unusual cause of small bowel obstruction, accounting for about 10% of cases. Most commonly, the small intestine is obstructed by extrinsic compression or local invasion by advanced gastro intestinal (Pancreatic, colonic, gastric) or

gynaecological (ovarian) malignancies. This mechanism accounted for 92% of neoplastic small bowel obstruction in a Mayo Clinic series. (23)

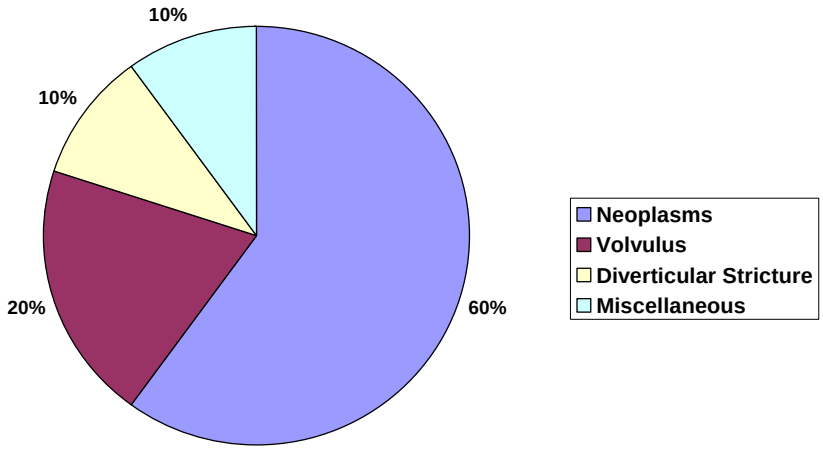
Hematogenous metastases from breast adeno carcinoma and melanoma may also involve the intestine with subsequent obstruction.

Primary neoplasms of the small intestine are the cause of obstruction in less than 3% of cases. Carcinoid tumors and adenocarcinoma have been variably reported as the most common malignancy of the small intestine. Adeno carcinoma of small intestine arises more frequently in the duodenum and jejunum than in ileum. (23)

a) Small bowel Obstruction



b) Colonic Obstruction



4. SIGMOID VOLVULUS

It is the commonest cause of large bowel obstruction in indigenous black Africans. The predisposing causes are long pelvic mesocolon, narrow attachment of pelvic mesocolon, overloaded pelvic colon, band of adhesions (peridiverticulitis), high residue diet and chronic constipation. Rotation nearly always occurs in an anticlockwise direction. The symptoms are of large bowel obstruction which may initially be intermittent, followed by the passage of large quantities of flatus and faeces. Constipation is absolute. Abdominal distension is an early and progressive sign. Vomiting occurs late. The classic appearance is of a dilated loop of bowel running diagonally across the abdomen from right to let with two fluid levels seen, one within each loop of bowel.

5. CROHN'S DISEASE

In crohn's disease, bowel obstruction usually occurs under two different conditions.

1. In the acute exacerbation of the disease, the bowel obstruction is the result, of a reversible inflammatory process within the bowel wall that causes a narrowing of the intestinal lumen.

2. Small bowel obstruction may also be the result of a chronic stricture.

6. RADIATION ENTERITIS (ENTEROPATHY)

Radiation injury to the bowel is a result of obliterative vasculitis and fibrosis consequent to radiation therapy. This progressive obliterative endarteritis is a dynamic, relentless process that may lead to a chronic recurrent low grade obstruction of the small intestine or cicatrization and bleeding in the colon and the rectum.

7. INTUSSUSCEPTION IN ADULTS

One segment of intestine (the intussusceptum) telescopes within an adjacent segment (the intussuscipiens), resulting in obstruction and possibly ischemic injury to the intussuscepting segment. In most adult patients (up to 93%) there is an underlying pathologic process, most commonly a tumor (benign in 52% and malignant in 48%). Congenital lesions (e.g: meckel's

diverticulum or small bowel duplications) or other localized abnormalities of the intestinal tract compose the remainder. Primary Idiopathic Intussuception accounts for only a small percentage of all instances in adults. (22)

8. OGILVIE'S SYNDROME (ACUTE COLONIC PSEUDO OBSTRUCTION)

This is a common and potentially dangerous condition. It presents with signs and symptoms and radiological appearances of large bowel obstruction but without any apparent mechanical cause. If left untreated, it can lead to ischemic necrosis and colonic perforation. The pathogenesis of this syndrome is unknown, but current evidence points towards an autonomic imbalance caused by Parasympathetic suppression. Motility enhancing drugs such as erythromycin have been effective in treating this condition. There are various reports of the success of neostigmine in achieving decompression.

PATHOPHYSIOLOGY

Absorption and Secretion

Intestinal obstruction has marked effects on intestinal absorption and secretion. In mechanical obstruction, intestinal fluid accumulates as the intestine distends, causing a major disruption in the orderly balance of secretion and reabsorption. In early intestinal obstruction (before 12 hrs) water and

electrolytes accumulate in the lumen because of a decrease in absorption with little effect on intestinal secretion.

After 24 hrs intraluminal water and electrolytes accumulate more rapidly secondary to a further decrease in absorption with a commensurate increase in intestinal secretion.

If obstruction persists, the proximal intestine dilates, absorption decreases, secretion increases, and net effect is intraluminal fluid accumulation.

Intraluminal fluid sequestration initiates an inflammatory cascade by attracting and activating neutrophils. The subsequent release of oxygen, free radicals and oedema promoting peroxidation products, such as leukotriene B4 and interleukin-1 exacerbates the plasma extravasation and transudation, resulting in a net fluid secretion into the obstructed bowel.

Blood Flow

When mechanical obstruction persists, intestinal distension increases, and intra luminal pressures increases, the effect on intestinal blood flow then becomes important, especially considering its association with strangulation obstruction.

Intestinal blood flow is related inversely to intraluminal pressures. When the intraluminal pressure is increased to 30 mm Hg, capillary streaming was interrupted; by 60 mm Hg, blood flow was interrupted in some vessels. (22)

As the obstruction persists other changes ensue. Reduced mucosal blood flow during small bowel obstruction leads to relative ischemia and hypoxia to villous tips. During this period, oxygen derived free radicals and their derivatives appear to play an essential role in the changes that follow the early phase.

Bacteriology

Normally mucosa of the gastrointestinal tract maintains a barrier to prevent resident bacteria that normally exist within the gut lumen from entering the systemic circulation. Under certain conditions, this immunologic gut barrier may break down. So indigenous bacteria colonizing the gastro intestinal tract can cross the mucosal epithelium (translocation) to infect mesenteric lymph nodes and even systemic organs.

Therefore with established mechanical small bowel obstruction, pre operative antibiotic prophylaxis is indicated and peritoneal soilage by spillage of small intestinal content must be avoided.

Systemic pathophysiologic effects of intestinal obstruction

Systemic response to intestinal obstruction involves an initial isotonic contraction dehydration secondary not only to the intestinal and peritoneal sequestration of extracellular fluid but also to the associated vomiting. Hypokalemia is secondary to the persistant vomiting, intestinal secretion, and

an increase in serum aldosterone concentration that occur in response to the hypovolemia. Cardiovascular effects manifest as tachycardia and relative hypotension secondary to hypovolemia.

Respiratory compromise may occur with severe abdominal distension or secondary to aspiration of vomitus. Intestinal infarction may precipitate a metabolic acidosis and sepsis with associated vascular collapse.

Causes of bowel distension

A. Gas : Originates from swallowed air produced within the intestine

B. Fluid : This consists of swallowed liquids and gastro intestinal tract secretions.

PATHOPHYSIOLOGY OF STRANGULATED BOWEL

OBSTRUCTION

Onset of intestinal obstruction

↓

Gas and fluid accumulates within the lumen of bowel

↓

Bowel distension

↓

Increased intra luminal and intra mural pressures

↓

Venous obstruction result in vascular engorgement, oedema and local venous hypertension

↓

Reflex arterial vasospasm

↓

Tissue hypoxia

↓

Intestinal Ischemia

↓

Bowel Infarction

The viability of the bowel is threatened by vascular compromise by

1. external compression (hernial orifices, adhesion, bands)

2. Interruption of the mesenteric flow (Vovulus, intussusception)

3. Primary obstruction of the intestinal circulation (mesenteric infarction)

Except in the case of mesenteric infarction, the venous return is affected before the arterial supply. The sudden increase in capillary pressure results in escape of intra vascular fluid and diapedesis of red blood cells into the bowel wall, its lumen and the herinal sac or peritoneal cavity. The bowel wall becomes oedematous and haemorrhagic. The peritoneal exudates becomes blood stained. As the arterial supply becomes jeopardized haemorrhagic infarction sets in. The mortality depends on the age and the extent of infarction and any delay in institution of appropriate treatment.

PARALYTIC OR ADYNAMIC ILEUS

Ileus refers to the failure of aboral passage of intestinal contents in the absence of mechanical obstruction.

Causes:

1. Laparotomy
2. Electrolyte derangements:

    e.g: Hypokalemia, Hyponatremia, Hypomagnesemia

3. Drugs:

Narcotics, Phenothiazines, Diltiazem, Anticholinergic agents

4. Intra abdominal inflammation:

Appendicitis, Diverticulitis, Perforated duodenal ulcer

5. Retroperitoneal inflammation or haemorrage

Lumbar compression fracture, Acute Pancreatitis, Pyelonephritis

6. Intestinal ischemia

Mesenteric arterial embolus (or) thrombosis, Mesenteric venous thrombosis, Chronic mesenteric Ischemia

7. Thoracic diseases

Lower rib fractures, Lower lobe pneumonia, Myocardial infarction

8. Systemic Sepsis

Post operative Ileus is thought to result from the loss of normal coordination of intestinal contraction by the intrinsic electrical activity of the bowel. (23)

CLINICAL FEATURES

The hallmark of intestinal obstruction is abdominal pain, vomiting, obstipation and abdominal distension.

The magnitude of symptoms depends on the degree of obstruction (i.e) complete or partial and the site and duration of obstruction.

BOWEL OBSTRUCTION: CLINICAL PRESENTATION AND RADIOLOGICAL FINDINGS

	Features	High small bowel obstruction	Low small bowel obstruction	Large bowel obstruction
1	Onset of symptoms	Sudden	Gradual	Insidious
2	Characteristics of pain	Epigastric, intense, colicky often relieved by vomiting, occasionally continuous	Periumbilical colic	Central or lower abdominal colic, possibly associated with generalised discomfort
3	Vomiting	Large volumes, bilious, frequent	Low volume and frequency; progressively faeculent with time	Intermittent, not frequent, faeculent
4	Tenderness	Epigastric or periumbilical usually mild unless strangulation is present	Diffuse and progressive	Diffuse
5	Distension	Absent	Diffuse and progressive	Diffuse
6	Obstipation	May or may not be present	Mild or moderate	Complete
7	Radiologic Findings	Abdomen may appear gasless or show distended proximal small bowel	Gaseous distension of small bowel, fluid levels on erect film	Gaseous distension of large bowel proximal to obstruction, small bowel may also show distension

CLINICAL PRESENTATION

1. Abdominal pain:

Pain is episodic, crampy and poorly localised. Simple obstruction provokes paroxyms of sharp pain, alternating with pain free intervals. If colickly pain becomes constant, strangulation or perforation may have developed. Unremitting pain, out of proportion to the physical findings is typical of mesenteric vascular ischemia.

2. Vomiting:

Vomiting occurs early in high small bowel obstruction but may be absent or develop late in distal small bowel obstruction or colonic obstruction. At first, the vomitus may contain altered food but later it becomes bile stained. Finally when the obstruction becomes complete and the dilated obstructed intestine allows bacteria to proliferate in the stagnant intestinal lumen the vomitus turns faeculent and foul smelling indicating late estabilished intestinal obstruction.

3. Obstipation:

Obstipation is a late feature. The failure to pass flatus or faeces, occurs after the bowel distal to the obstructed segment empties. Partial bowel obstruction often permits passage of small bouts of gas and faeces.

4. Abdominal distension:

It is usually delayed with obstruction of the most proximal small bowel. Gross distension is obvious in sigmoid volvulus, Hirschprung's disease and low small bowel obstructions and caecal neoplasms.

PHYSICAL EXAMINATION

1. Patients usually appear ill.

2. Signs of dehydration – sunken eyes, dry mucous membranes, loss of skin turgor, tachycardia and hypotension

3. Fever – suggests the possibility of strangulation or peritonitis.

4. Abdomen is usually distended.

5. Visible intestinal peristalsis, (or) colonic peristalsis are sometimes visible through the abdominal wall of these patients.

6. Surgical scars should be noted. Because of the etiologic implication of previous operations for e.g: the presence of adhesions, cancer or Crohn's disease.

7. Palpation of the abdomen in simple obstruction may reveal minimal tenderness.

8. Muscle guarding may be elicited during attacks of colic.

9. Marked tenderness, peritoneal signs, or a palpable mass suggests a closed loop obstruction or strangulation.

10. Per rectal examination – may reveal blood, a mass, or a Blummer shelf nodule.

11. Per Vaginal examination – might yield evidence of gynaecologic malignancy or an inflammatory lesion.

12. Auscultation:

    - High pitched tinkling bowel sounds (Borborygmi) heard in early obstruction.

    - Progressive distention in a late phase of obstruction inhibits the smooth muscle contraction, peristalsis ceases, and the abdomen becomes relatively quiet.

    - Strangulation and peritonitis are marked by a silent abdomen

13. In case of adynamic ileus, the abdomen is often distended, bowel sounds are scattered without peristaltic rushes and pain and tenderness are usually minimal. (4)

DIAGNOSIS

Laboratory Test

The diagnosis of bowel obstruction is not dependent on any set of laboratory findings. More over, blood tests are not helpful in differentiating simple from strangulated obstruction.

Leukocytosis of greater than 18,000 cells / cmm is suggestive but not confirmatory of strangulation which can also present with leucopenia.

Metabolic acidosis, amylase activity, serum phosphate level, creatine kinase, lactate dehydrogenase activity, liver enzyme activity and hematocrit are unreliable predictors of strangulation.

Elevated serum amylase activity may be due to

1. A reflux of pancreatic amylase resulting from back pressure from the duodenum.

2. Leakage of intraluminal amylase into the peritoneum with subsequent absorption.

3. Release of intestinal amylase from compromised bowel.

The presence of a significant metabolic acidosis is an ominous sign, yet its sensitivity as an indicator of strangulation is only 75%. Some patients with dead or dying bowel have no base deficit. (4)

Creatine kinase determination may be somewhat useful, as it is elevated in 71% of patients with strangulated bowel. (4)

Initial fluid shift results in contraction of the extracellular fluid with antidiuresis, renal reabsorption of sodium, a rising blood urea nitrogen level and an increased urine specific gravity.

With progressive obstruction and vomiting patients tend to develop hyponatraemia, hypokalemia, metabolic acidosis, hypochloremia and uremia.(4)

RADIOGRAPHIC CONFIGURATIONS

Usually the diagnosis of bowel obstruction is first suggested by the history and the physical examination. However, radiologic investigations are invaluable, not only in confirming or refuting the diagnosis, but also in determining if the obstruction is high or low, is partial or complete and involves small bowel or large bowel.

Plain Films

On plain supine and upright radiographs of the abdomen, the cardinal findings that suggest the diagnosis of small bowel obstruction reflect the

1. Accumulation of air and fluid proximal to the point of obstruction.

2. Clearance or absence of fluid and air distal to the obstruction.

3. Dilated loops of small intestine.

Dilated loops of small intestine are defined as those larger than 3 cms in diameter. The proximal colon is considered dilated when the diameter reaches 8 to 10 cms and sigmoid colon 4 to 5 cms. (4)

The extent of intestinal dilatation is a function not only of the site of obstruction but also of the degree and duration of obstruction. Abdominal radiographs fail to show any abnormal findings in as many as 20% of patients with proximal intestinal obstruction, especially if the patient has recently vomited and emptied the stomach. (4)

Different parts of the bowel have charecteristic features as follows:

Jejunum	-	Valvulae conniventes are seen
		Concertina / ladder effect
Ileum	-	Featureless (wangensteen)

Caecum	-	Rounded gas shadow in right iliac fossa
Large bowel	-	Haustral folds
Sigmoid volvulus	-	Bent inner tube appearance / coffee bean appearance

Small bowel pattern occupies the more central portions of the abdomen whereas the colonic shadow is on the periphery of the abdominal film or in the pelvis.

The plain radiograph may show air in the biliary tree (Pneumobilia) indicative of a biliary enteric fistula. When small bowel obstruction coexists this spectrum of findings is almost pathognomonic of gallstone Ileus.

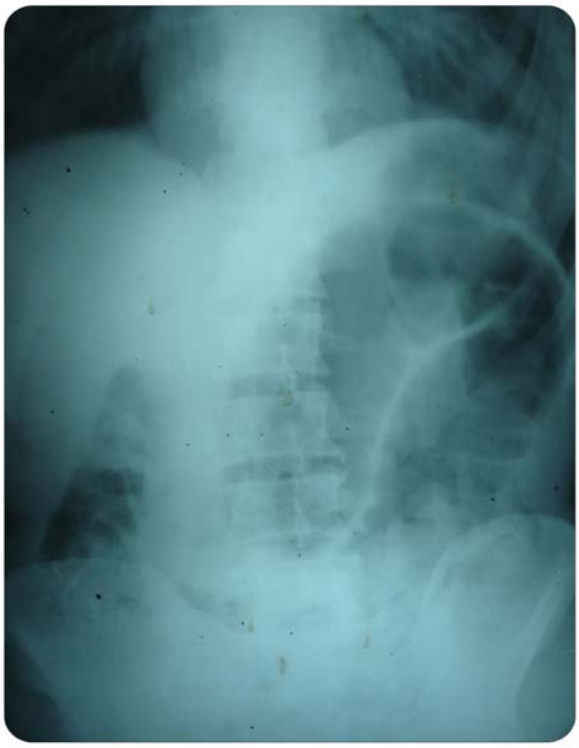
Signs of strangulation are

(i) A fixed loop of bowel that remains stationary in sequential films.

(ii) Pseudotumour sign: a fluid filled gangrenous loop appearing as a soft tissue mass.

(iii) Pneumatosis Intestinalis.

SIGMOID VOLVULUS



DILATED BOWEL LOOPS

CONTRAST STUDIES

Contrast studies of the gastro intestinal tract either from above, as with small bowel follow through or enteroclysis or from below with contrast enemas may be helpful.

These studies can provide specific localisation of the point of obstruction and may identify the nature of the underlying lesion. It can be useful in differentiating mechanical obstruction from adynamic Ileus.

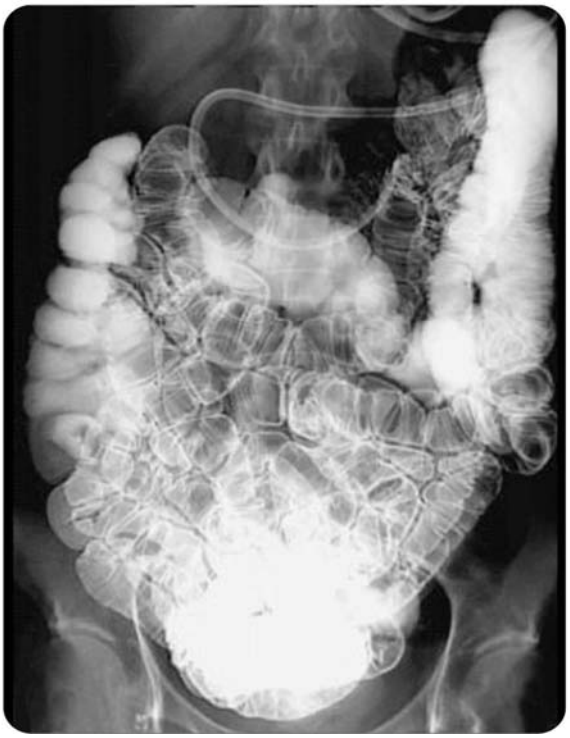
The most definitive studies are obtained by infusing barium through a tube in the distal duodenum, a procedure known as enteroclysis.

Barium is reported to cause impaction but does not occur in small bowel obstruction though it can convert obstruction in the colon. Barium provides better resolution and causes less cramping and vomiting. Water soluble contrast (gastrograffin) is used in case of perforation. It is hyperosmolar and inadvertent aspiration can be lethal.

Contrast enemas provide important information in large bowel obstruction where the exact site and cause of obstruction is unclear. Contrast enema performed under carefully controlled conditions (dilute barium, hydrostatic pressures of less than 80 mm Hg) may aid in planning therapy. Another situation in which a contrast enema is of potential benefit is very distal small bowel obstruction.

MULTIPLE AIR FLUID LEVELS

ENTEROCLYSIS



COMPUTED TOMOGRAPHY

80-90% Sensitive

70-90% Specific

1. Finding of small bowel obstruction:

Discrete transition zone with dilatation of bowel proximally, decompression of bowel distally, intraluminal contrast that does not pass beyond the transition zone and a colon containing little gas or fluid.

2. Closed loop obstruction is suggested by the presence of a ‘U’ shaped or ‘C’ shaped dilated bowel loop associated with a radial distribution of mesenteric vessels converging towards a torsion point. (22)

3. Bowel wall thickening, pneumatosis intestinales, inflammatory changes and haemorrhage in the mesentry and poor uptake of intra venous contrast into the wall of the affected bowel in cases of strangulation.

4. In-case of cancer, it is useful for staging the disease.

ULTRASONOGRAPHY

Abdominal ultrasound has also been proposed as an aid in the diagnosis of obstruction of the small bowel and may identify both its location and etiology. Ultrasound can delineate extra luminal masses involved with areas of

intestinal dilation, more over real time ultrasonography examining blood flow can aid in the diagnosis of strangulation obstruction, where its accuracy may be as high as 90%. (22)

SPECIAL PROCEDURES

1. ENDOSCOPY:

Can reveal obstructing lesions at both proximal and distal ends of the alimentary tract, but despite recent advances, enteroscopy is less efficient in the mesenteric small intestine.

2. PARACENTESIS:

Paracentesis and assay of peritoneal fluid may be of value when the clinical picture is confusing. A finding of white blood cells or bacteria indicates strangulation and would support immediate laparotomy.

3. LAPAROSCOPY:

Laparoscopy can differentiate simple from strangulated obstruction and can occasionally be therapeutic as well. When constricting adhesions are accessible and not too dense, laparoscopic lysis may be feasible.

Recent trends suggest that scanning techniques can be useful in detecting strangulation. In rabbits and dogs, radionuclide scanning with

technetium diphosphonate showed increased uptake of Isotopes by the Ischemic bowel. Xenon – 133 also has been used to detect early strangulation induced in experimental animals. When instilled intraperitoneally, ^{133} Xe rapidly disappears from normal animals but there is a significant delay in strangulated obstruction. (4)

MANAGEMENT

The fundamental principles involved in managing patients with obstruction are

1. Resuscitation by correction of hemodynamic and electrolyte imbalance.

2. Decompression of the gastro intestinal tract.

3. Timely surgical intervention.

All patients with suspected strangulation require emergency laparotomy: some patients with simple obstruction may, in certain circumstances be handled conservatively.

(i) Fluid and electrolyte repletion:

Fluid and electrolyte deficits should be rapidly restored in all patients, however when strangulation is suspected surgical intervention should not be delayed while awaiting complete normalisation. Surgical repair of the injury is

resuscitating in itself. Fluids are lost not only externally but also by third space sequestration. The magnitude of these losses cannot be directly measured, and therefore, the adequacy of replacement must be assessed by clinical signs, assisted by central line monitoring, pulse rate, blood pressure and urine output.

In the elderly and in patients suspected of harbouring gangrenous bowel, a Swan Ganz catheter is essential to record pulmonary capillary wedge pressure, cardiac output, and mixed venous oxygen saturation. Changes in blood composition take much longer to correct than do volume losses. Profound hypokalaemia, may require up to 24 hours to reverse.

Serum electrolyte and acid base determinations help guide the choice of fluids to be administered. If gastric acid loss predominates, normal saline is used, but for most bowel obstructions, lactated Ringer's solution is more appropriate.

Intra vascular volume is restored with crystalloid solutions, the hematocrit can be expected to drop with strangulation. A significant amount of blood may be lost into the bowel and peritoneal cavity and this should be replaced by packed cell transfusion as needed.

(ii) Intubation:

All patients with obstruction require aspiration of gastro intestinal contents, through either a short or long tube.

The most popular tubes are nasogastric tubes.

Nasogartric tubes help by,

1. Decompressing the stomach and the most proximal intestine.

2. Preventing distension by swallowed air.

3. Minimizing the risk of aspiration during induction of anaesthesia.

Tubes are either long (e.g: miller abbot) or short (non vented Ryle's and vented salem). Long tubes like miller abbot are of the double type with a channel for aspiration and with a balloon at the other end for advancement.

Long tubes may be useful initially to treat small bowel obstruction, particularly when the nasogastric suction has failed; intra operatively to decompress more distal bowel and to identify points of intrinsic obstruction and postoperatively to stent the bowel.

ANTIBIOTICS

Antibiotics do not alter the course of simple obstruction but are administered preoperatively because of the possibility of inadvertent enterostomy and attendant peritoneal soilage, which might result in intra abdominal abscess or wound infection.

In cases of suspected or known strangulation, potentially pathogenic translocated bacteria and toxins can be life threatening and antibiotic therapy is essential.

Antibiotics selection should be from among those effective against both aerobic gram negative bacilli and anerobes such as bacteroides. A reasonable choice in simple obstruction might be a second generation cephalosporin such as cefoxitin or cefotetan or a synthetic penicillin, in combination with β lactamase inhibitor (eg: ampicillin and sulbactam). Because of the life threatening sepsis that occurs in strangulation, it may be advisable to add an aminoglycoside, such as gentamicin.

Monoclonal antibodies directed against endotoxins from gram negative bacteria have been developed. HA-1A and E5 are examples of immunoglobulin M antibodies used in current trials in patients experiencing shock due to complicated bowl obstruction. (4)

ROLE OF EXPECTANT NON OPERATIVE MANAGEMENT

Surgery may be delayed under certain circumstances.

1. Post operative obstruction

Distension, vomiting and failure to pass flatus early after abdominal surgery are usually due to transient (physiologic) ileus, but mechanical

obstruction can also follow intra abdominal operations. However overlooked strangulation remains a potential risk.

2. Later Post operative obstruction

A special situation involves small bowel obstruction that develops 10 days to 4 weeks after a previous celiotomy. This window of time represents the most dangerous time for reoperation because the adhesions forming from the previous celiotomy can be extremely thick, intense and vascular. Strong consideration should be given to a primary non-operative approach with a planned gastric decompression for 3 to 6 months (by a percutaneous endoscopic gastrostomy or even a tube pharyngostomy) and parenteral nutritional support. (22)

3. Intussusception

Infants with ileocaecal intussusception usually respond to a controlled hydrostatic reduction of the intussusception, which avoids operation entirely.

4. Sigmoid Volvulus

In patients with sigmoid volvulus sigmoidoscopy or colonoscopy can achieve decompression of the obstructed sigmoid loop.

5. Crohn's disease

In patients with intestinal obstruction due to an acute exacerbation of crohn's disease, a period of conservative medical treatment with systemic steroids or other anti inflammatory agents may lead to resolution of the obstruction and prevent the need for operative intervention.

6. Partial Obstruction

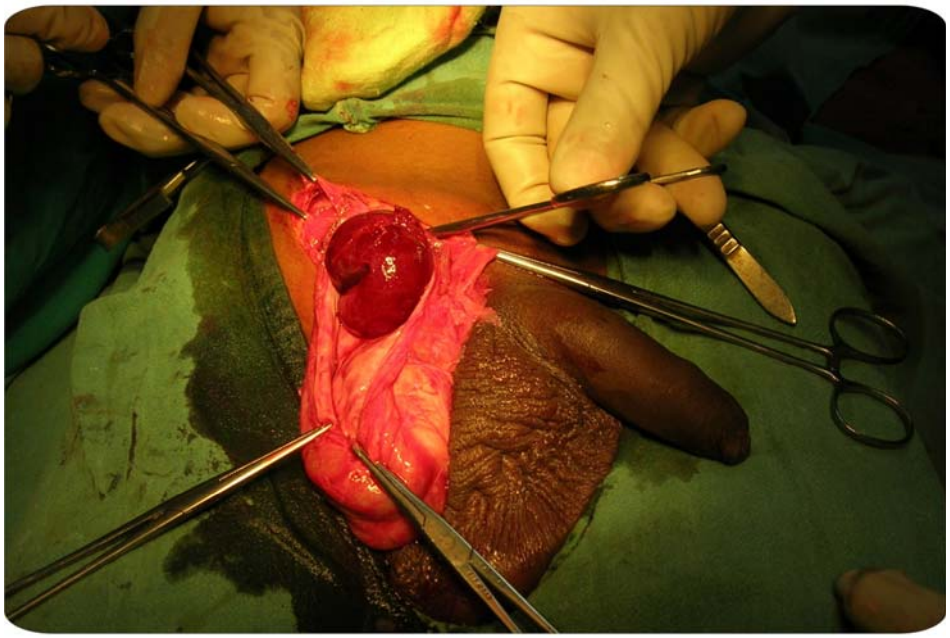
Patients with partial small bowel obstruction may be managed by less urgent operative treatment than patients with acute complete obstruction. If a patient has already undergone multiple operations for obstruction due to adhesions, further operation may carry little prospect of success and can be hazardous if the bowel is opened inadvertently.

Conservative Therapy is Contra Indicated in

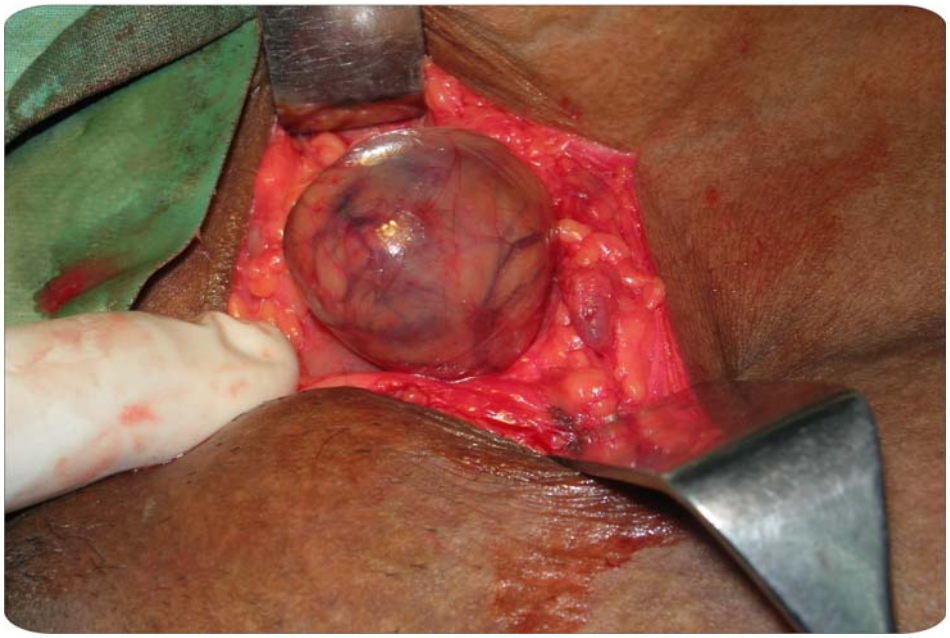
1. Suspected strangulation
2. Closed loop obstruction
3. hernia complicated by obstruction
4. Small bowel obstruction without an abdominal scar or hernia

OPERATIVE PICTURES

OBSTRUCTED INGUINAL HERNIA



OBSTRUCTED FEMORAL HERNIA



SURGICAL MANAGEMENT

The most critical decision in managing obstruction is distinguishing between simple and strangulated obstruction. A common saying is the “sun should never rise and set on a complete small bowel obstruction”.

There is no single clinical sign (or) diagnostic test that accurately diagnoses strangulation. Rarely does a patient exhibit all the signs of strangulation viz.,

Fever

Tachycardia

Localised abdominal pain

Rebound Tenderness

Guarding / rigidity

Leukocytosis

However, in the absence of all these signs, dead on dying bowel is found in less than 10% of cases. No improvement by 48 hours or clinical deterioration at any time mandates emergency surgery. (4)

When the exact cause of the obstruction is unknown, an appropriate incision, most often midline is essential. It is necessary to determine the

1. Site of obstruction

2. Nature of Obstruction

3. Bowel viability

The cecum is the best guide to the site of obstruction. If collapsed, the obstructive lesion lies proximally and if distended, then a colonic lesion is likely. From the ileocaecal junction the small bowel is traced upwards following the collapsed loop to the junction. With the distended loops where the obstructive lesions should lie.

The assessment of bowel viability may sometimes be difficult. The problem that the surgeon faces is,

1. determining the viability in borderline cases

2. delineating the extent of involvement. Obviously infarcted bowel is

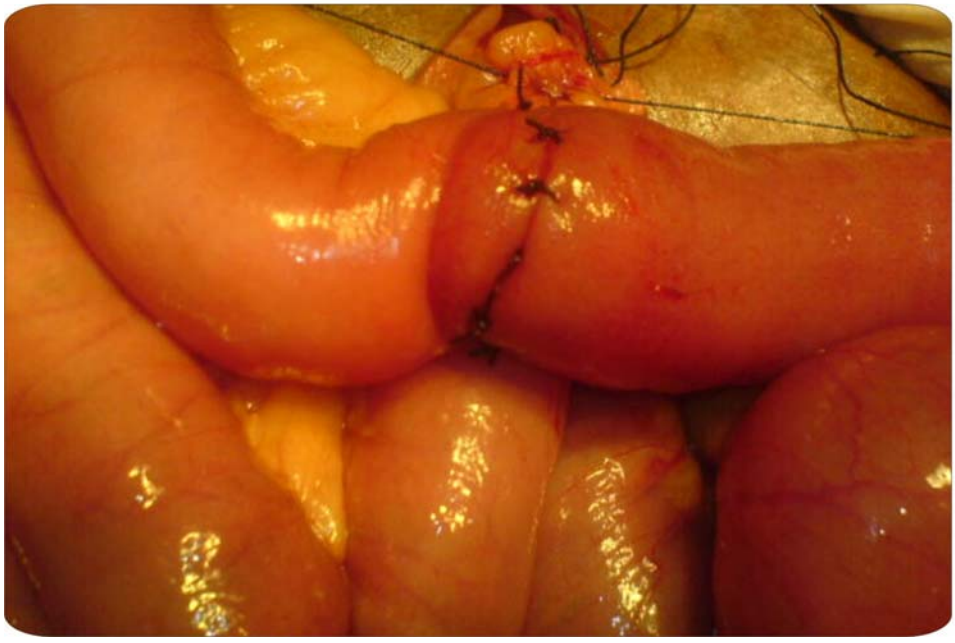
    a) greenish black in appearance

    b) with no peristalsis and complete atony

    c) with absence of bleeding from cut surfaces

SMALL BOWEL GANGRENE

RESECTION AND ANASTOMOSIS OF SMALL BOWEL



d) with absent pulsations in the vasa recta and mesenteric vessels.

Intra operative methods of determining the viability of the bowel include the use of Doppler flow to measure blood flow in the anti mesenteric border, and injecting intravenous fluorescein and examining the bowel with woods lamp.

When the bowel viability is undeterminable then it would be safer to exteriorize both ends. This allows daily inspection of the circulatory status of the exteriorized ends. When doubt remains regarding a segment of a bowel, a second look operation may be carried out 24 hours later. (4)

The nature of surgical procedure depends on the cause

1. Adhesions – Adhesiolysis

2. Obstructed hernia – Reduction of the herniated bowel and closure of the defect.

3. Malignant tumour of small bowel: if feasible, primary resection and anastomosis can be done. Otherwise a simple bypass to relieve the obstruction as an emergency procedure.

4. Inflammatory bowel disease: Bowel resection and (or) stricturoplasty if non operative treatment fails.

5. Intra abdominal abscess: CT guided drainage is usually sufficient to relieve obstruction.

6. Radiation enteritis: Possible resection of irradiated bowel or bypass of the affected area

7. Intussusception:

   < 24 hrs – hydrostatic reduction

   > 24 hrs – Resection and anastomosis

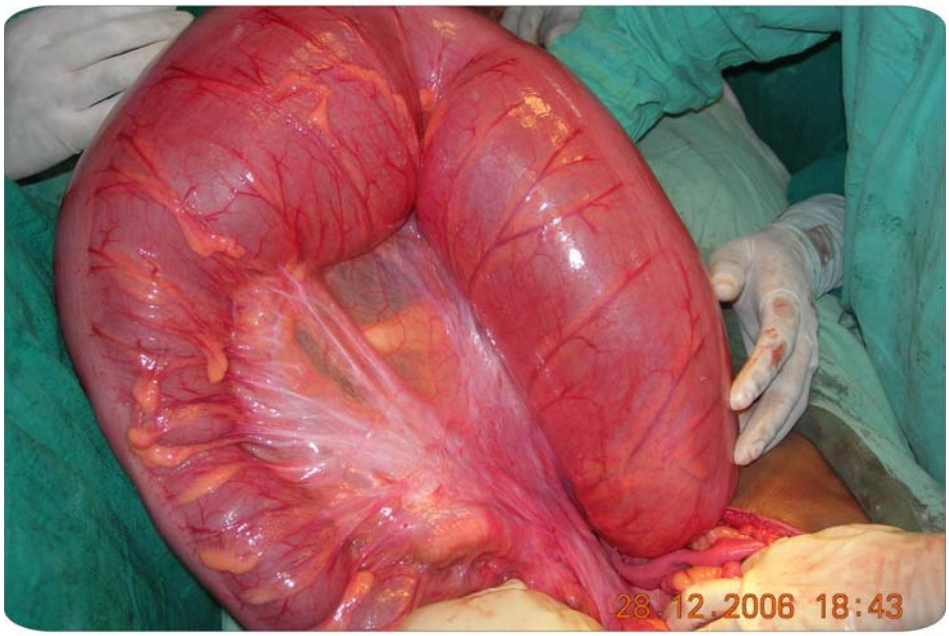
8. Recurrent intestinal obstruction

It is usually due to adhesions. Adhesiolysis is difficult, hence Plication operation is indicated. Anti adhesive agents like 5% polyethylene glycol can be used.

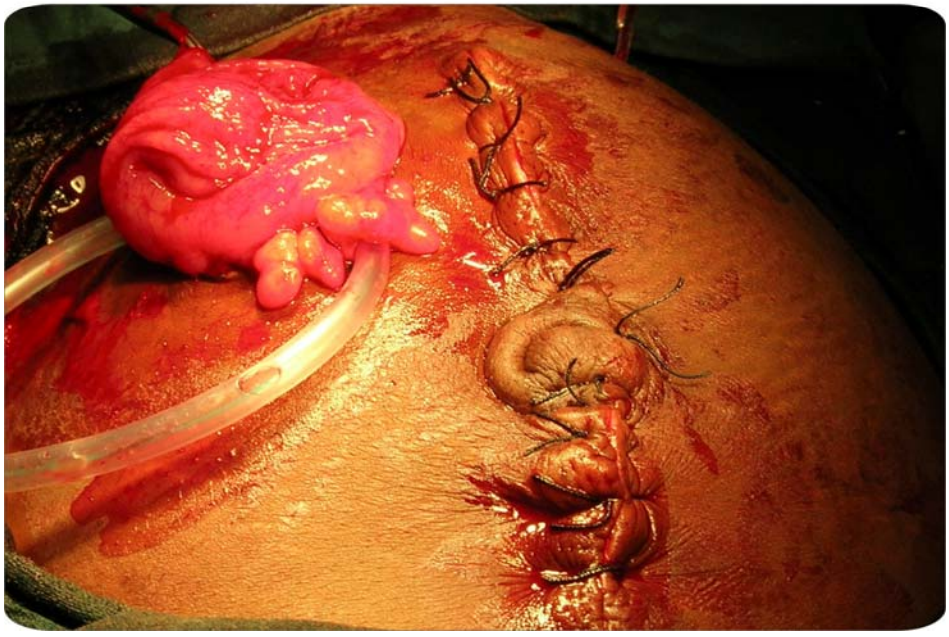
9. Sigmoid Volvulus:

Sigmoidectomy. Intestinal continuity may be restored by end to end (or) end to side anastomosis using stapler or hand sewn technique.

SIGMOID VOLVULUS



COLOSTOMY



10. Caecal volvulus:

Caecopexy (Suturing the right colon to the right paracolic gutter) and caecostomy which has an added advantage of decompressing the bowel in addition to fixation.

If the bowel is gangrenous, resection in the form of right hemicolectomy is necessary.

11. Colonic cancer

Right sided colonic obstruction

Obstructive right sided colonic tumours can be treated by a primary resection and anastomosis. Goligher and Smiddy were the pioneers who described right hemicolectomy as the procedure of choice for obstructing tumours of the right colon. Intestinal continuity may be restored by side to side stapled ileo transverse anastomosis using a linear cutter stapler (multifire GIA 80, Autosuture) or by handsewn technique.

When the colon is obstructed by a carcinoma of the splenic flexure, an extended right hemicolectomy is the procedure of choice with anastomosis between the ileum and descending colon.

Left sided colonic obstruction

Left sided colonic obstructions were treated by three stage procedure consisting of a primary colostomy, subsequent definitive resection and finally reversal of the colostomy. This three stage procedure was gradually replaced by a two stage (Hartmann's procedure) and more recently by single stage procedure. In favourable circumstances a single stage procedure is a safe option in the management of malignant left sided colonic obstruction.

Single stage procedure either in the form of a subtotal colectomy with primary ileosigmoid or ileorectal anastomoses or an intra operative colonic lavage with immediate colo colonic or colo rectal anastomosis can be done.

The SCOTIA (subtotal colectomy versus on table irrigation with anastomosis) study group performed the first randomized trial comparing these techniques for the management of left sided malignant colonic obstruction. Hospital mortality rates were similar in the two groups, post operative morbidity was somewhat higher in the subtotal colectomy group.

The SCOTIA study group concluded that segmental resection following intra operative irrigation was the preferred option except in cases of perforation of the caecum when subtotal colectomy was more appropriate.

RECENT ADVANCES

The use of self expandable metallic stents (SEMS) in relieving left sided colonic obstruction has become popular. The use of SEMS in colonic obstruction was first reported by Itabashi in 1993.

SEMS are effective in relieving colonic obstruction temporarily in preparation for subsequent surgery, hence avoiding an operation in the emergency setting. Patients can subsequently undergo a single staged operation and can avoid a stoma in favourable circumstances. SEMS also provide a good means of palliating unresertable obstructing tumours.

Tejero, in 1997, introduced a three staged procedure for the relief of malignant left sided colonic obstruction

1. resolution of the obstruction by means of a stent placed at the site of tumour

2. recovery of the general state of the patient.

3. elective and final surgery (if not suitable, the stent may be used as a definitive palliative treatment). (12)

ROLE OF LAPARASCOPY IN THE MANAGEMENT OF BOWEL OBSTRUCTION

Today, laparoscopy is being applied to a number of obstructing small bowel and colonic processes, such as relief of small bowel obstruction secondary to adhesions, foreign body removal, bypass procedures for malignant and benign disease causing intestinal obstruction, resection for appendicitis or a meckel's diverticulum causing intestinal obstruction and evaluation and resection for inflammatory bowel disease (ie. crohn's disease).

Laparoscopic treatment of small bowel obstruction if successful leads to a shorter hospital stay and quicker rehabilitation of the patient and has good long term results. With further experience, laparoscopic exploration is an excellent diagnostic modality in acute small bowel obstruction and may allow a fully laparoscopic adhesiolysis with relief of the intestinal obstruction.

Patients with complete and or distal small bowel obstruction are not candidates for laparascopic management. (22)

Conclusion

In conclusion, most of the recent advances in the management of bowel obstruction consist of developments in the imaging modalities available to assist in the diagnosis itself, particularly with regard to the distinction between partial and complete obstruction. Unfortunately little progress has been made

to enable physicians to detect early reversible strangulation. Because of the inability to detect reversible ischemia, there is a substantial risk of progression to irreversible ischemia when surgery is delayed for an extended period of time, especially in the setting of suspected complete obstruction.

It is encouraging, that some advances have been made in understanding the patho physiology and prevention of adhesion formation. Research efforts in the future should continue to focus on these issues as well as on the development of methods to better recognize early signs of strangulation.

MATERIALS AND METHODS

This is a prospective study of 100 patients with acute intestinal obstruction admitted in the Department of General Surgery at the Government Kilpauk medical college and hospital from May 2005 to May 2007.

From the time of presentation the cases were followed up till the time of discharge / death. X ray abdomen erect was routinely taken for all patients.

The details of symptoms at presentation, the diagnosis entertained, investigations reports, type of surgery and outcome have all been recorded and tabulated in a master chart for reference and comparison. The final outcome has been sub grouped based on the degree of incapacitation to death.

The observations were compared with the current literature on acute intestinal obstruction and conclusions were drawn.

OBSERVATIONS

A total of 100 cases of acute intestinal obstruction were included in the study from May 2005 to May 2007. After admission all the cases were followed up and the relevant details were recorded. This was summarized into a master chart. The comparable tabulations permit certain statistical inferences to be made that are presented below.

1. Age and Sex Incidence

Age Distribution

There is no age bar for the occurrence of acute intestinal obstruction

Table 1

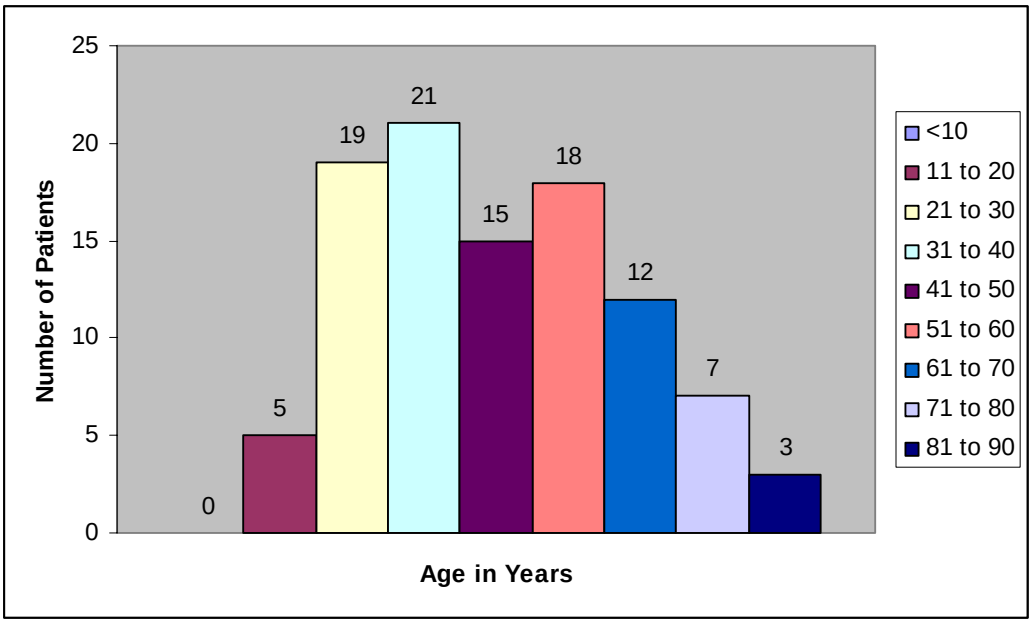
Age in years	No. of Patients
0-10	0
11-20	5
21-30	19
31-40	21
41-50	15
51-60	18
61-70	12
71-80	7
81-90	3

The most common age group to be affected was 31-40 years.

Average age of presentation of acute intestinal obstruction was 45.18 years

Diagram 1

Age Distribution



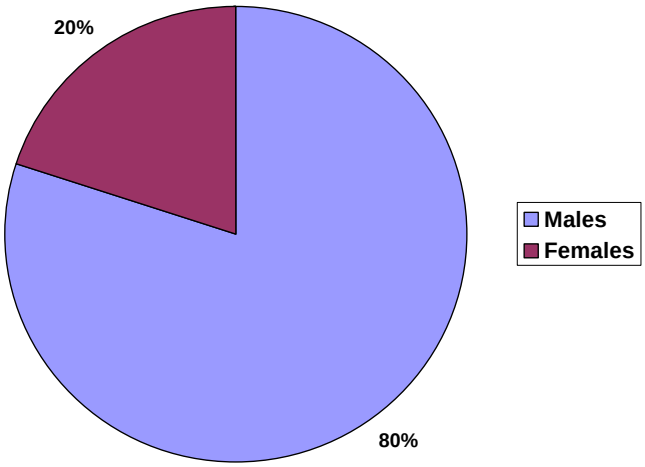
Sex Distribution

The sex distribution of Acute Intestinal obstruction is tabulated below

Table 2

Sex	Number of cases	Percentage
Males	80	80
Females	20	20

Diagram 2



There is a male preponderance seen in acute intestinal obstruction.

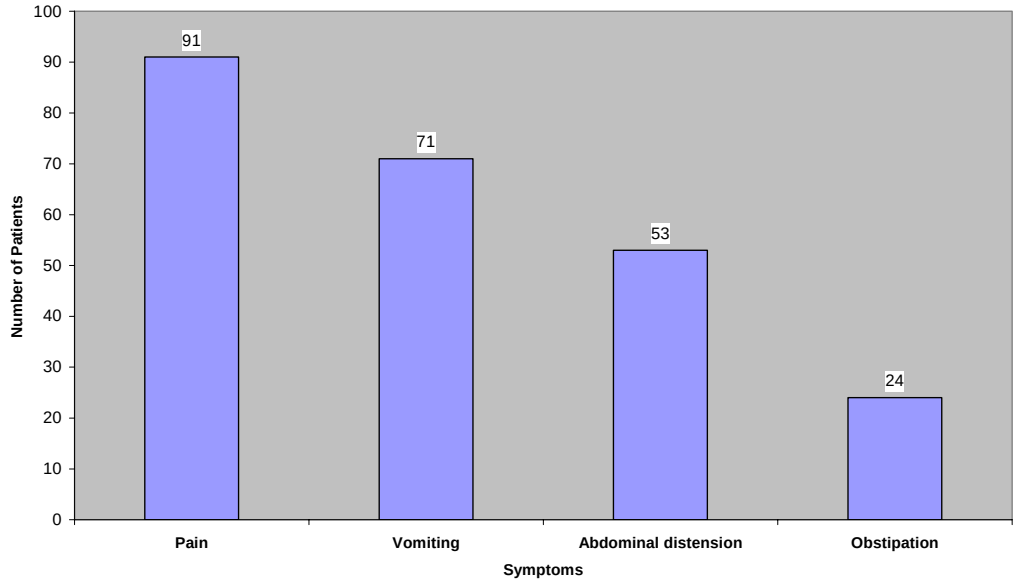
The male:female ratio is 4:1

2. Clinical symptomatology

Table 3

Symptoms	No. of cases	Percentage
Abdominal pain	91	91
Vomiting	71	71
Abdominal distension	53	53
Obstipation	24	24

Diagram 3



Abdominal pain was present in majority of the cases. The intensity of pain was graded from mild to severe.

The severity of vomiting was graded from mild (+) defined as bouts less than 5 per day, moderate (++) >5 bouts per day to severe (+++) ie. frequent bilious vomiting. Vomiting was present in 71 cases.

Abdominal distension was present in 53 cases. The absence of distension in 47 cases could be due to early presentation.

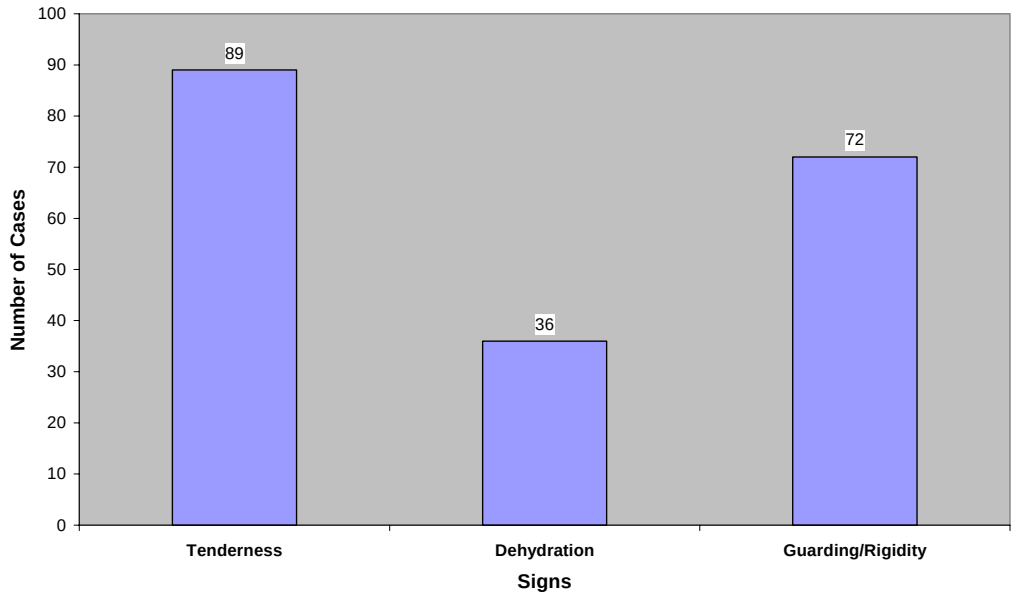
24 patients presented with obstipation.

Signs

Table 4

Signs	No. of cases	Percentage
Tenderness	89	89
Guarding / rigidity	72	72
Dehydration	36	36

Diagram 4



Tenderness was present in 89 cases, guarding and rigidity was elicited in 72 cases. 36 cases presented with signs of dehydration viz., dry tongue, sunken eyes, tachycardia, oliguria and hypotension.

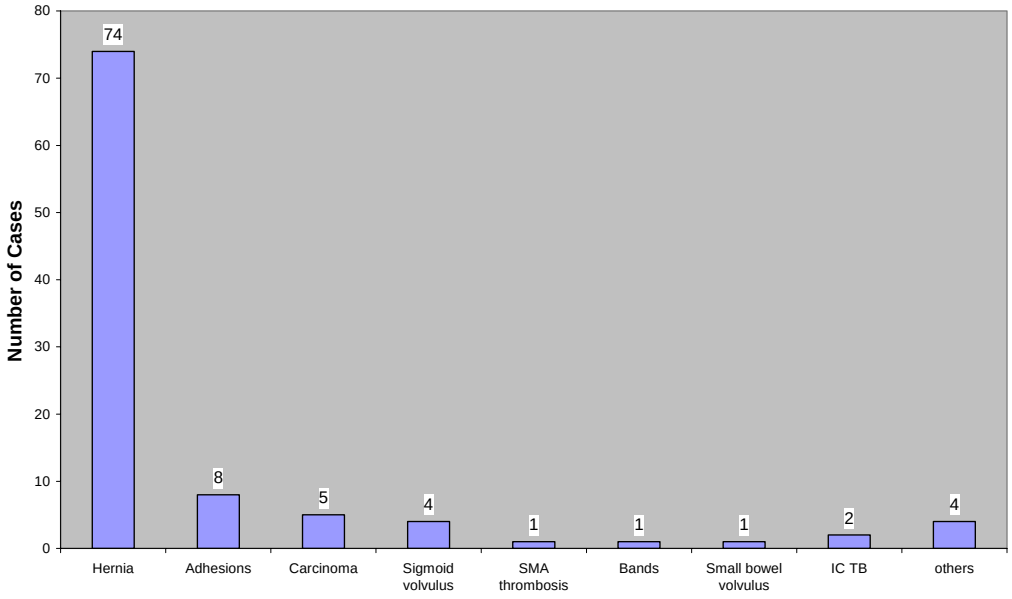
3. Disease Spectrum

Hernia was the commenest cause of acute intestinal obstruction. It accounts for 74% of the cases.

Table 5

Etiology	No. of Cases	Per centage
Obstructed Hernia	74	74
Adhesions	11	11
Carcinoma large bowel	5	5
Sigmoid Volvulus	4	4
Small bowel volvulus	2	2
Ileo caecal tuberculosis	2	2
Superior mesenteric artery thrombrosis (ischemic stricture)	1	1
Bands (Ladd's)	1	1

Diagram 5



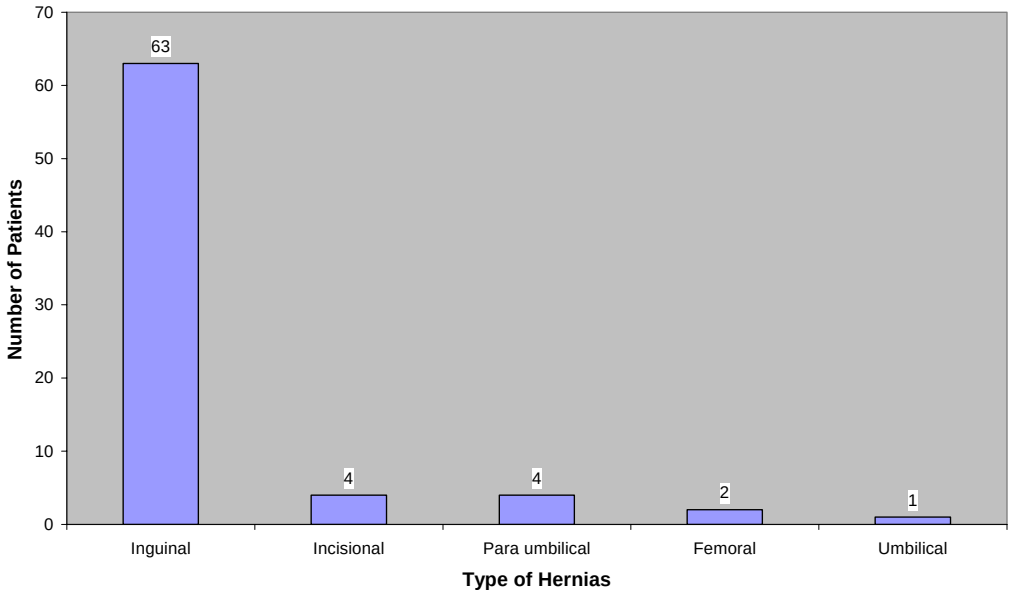
Adhesive obstruction was present in 12 cases, carcinoma large bowel 5 cases, sigmoid volvulus 4 cases, Ileocaecal TB 2 cases, superior mesenteric artery thrombosis, Ladd's band, small bowel volvulus and others accounting for 6 cases.

4. Types of hernia responsible for Acute intestinal obstruction was as follows

Table 6

Type	No. of Cases	Percentage
Inguinal	63	63
Incisional	4	4
Paraumbilical	4	4
Femoral	2	2
Umbilical	1	1

Diagram 6



In this study, Inguinal hernias were found to be most important cause for acute intestinal obstruction.

Strangulation of the bowel occurred in 7 cases and the causes were

Table 7

Etiology	No. of cases	Percentage
Inguinal Hernia	5	5
Incisional Hernia	2	2

6. Investigations

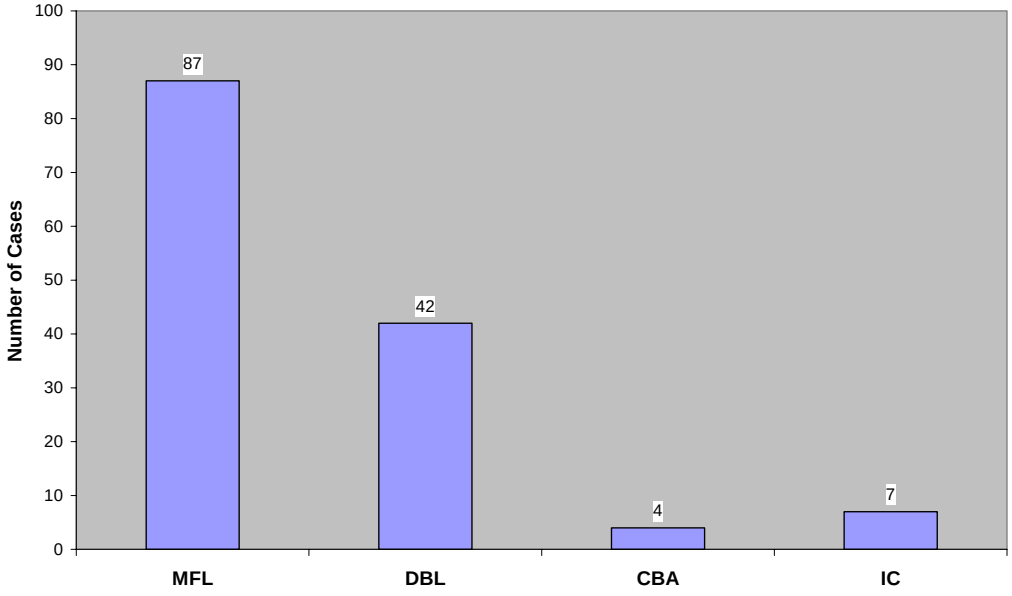
On X-Ray abdomen the commonest findings was of a multiple fluid levels seen in 87 cases, dilated bowel loops seen in 42 cases and 4 cases presented with classical coffee bean appearance.

Table 8

Findings	No. of cases	Percentage
Multiple fluid levels (MFL)	87	87
Dilated bowel loops (DBL)	42	42
Coffee bean Appearance (CBA)	4	4
Inconclusive (IC)	7	7

7 cases had fluid levels less than 3 and were considered as inconclusive

Diagram 8



Ultrasonogram

Table 9

Findings	No. of cases	Percentage
Dilated Bowel Loops	42	42
Free Fluid	15	15
Normal / Inconclusive	42	42

43 cases show dilated bowel loops.

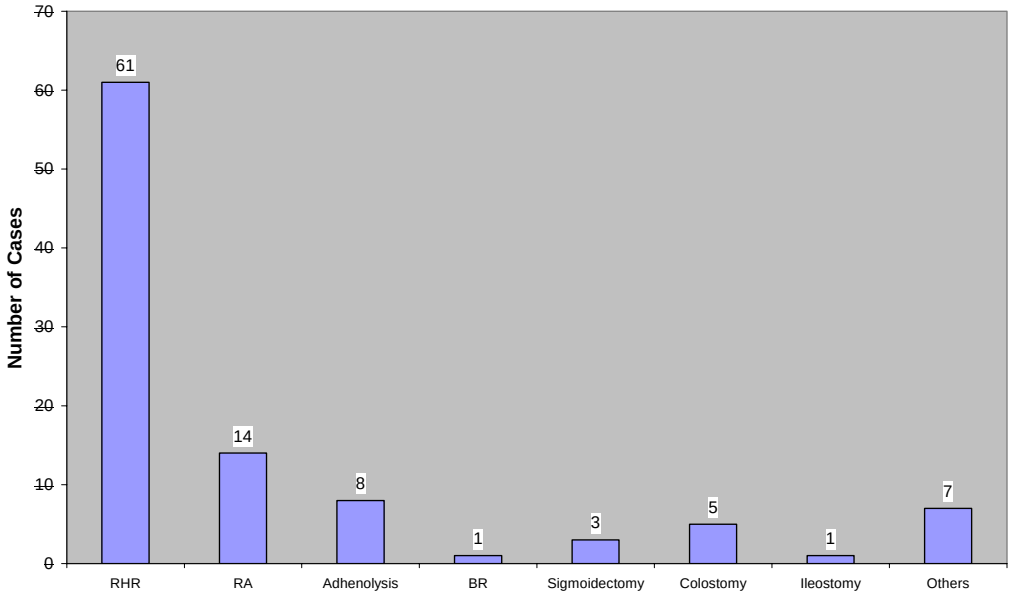
Free fluid is seen in 15 cases

6. Treatment

Table 10

Procedure	No. of Cases	Percentage
Reduction and hernia repair (RHR)	61	61
Resection and anastomosis (RA)	14	14
Adhesiolysis	8	8
Colostomy	5	5
Sigmoidectomy	3	3
Band Release (BR)	1	1
Ileostomy	1	1
Others	7	7

Diagram 9



7. Outcome

The outcome was graded into 4 classes

Grade I was an uneventful recovery

Grade II was with minor morbidity

Grade III was with major morbidity

Grade IV was death post operatively

Minor morbidity: Was defined as wound infertion, mild cardio pulmonary deficit, post operative ileus and self limiting intestinal leak

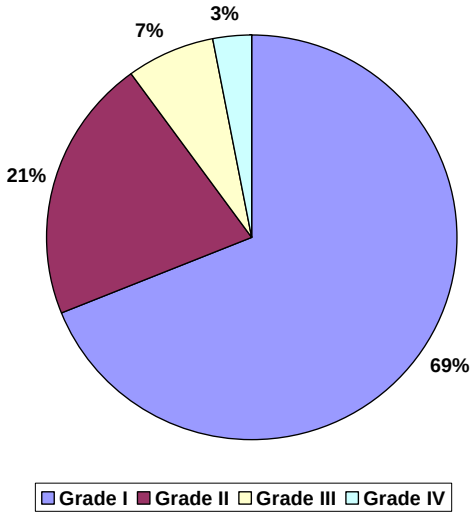
Major morbidity: was defined as wound dehiscence, moderate to severe cardio pulmonary deficit, enterocutaneous fistula.

Table 11

Outcome	No. of Cases	Percentage
Grade I	69	69
Grade II	21	21
Grade III	7	7
Grade IV	3	3

Majority of the patient had an uneventful recovery (69%). Death occurred only in 3 cases (3%).

Diagram 10



DISCUSSION

In the study of acute intestinal obstruction which was carried out for a period of 2 years (between May 2005 and May 2007), in the Government Kilpauk Medical College and Hospital, 100 cases of acute intestinal obstruction were studied based on clinical evaluation and radiographic evidence. All the patients were followed up during the hospital stay and the relevant details were catalogued in the master chart for comparison and for drawing statistical inferences.

AGE AND SEX INCIDENCE

The commonest age group affected was between 31 and 40 years (21 persons). The next common age group was between 51-60 years (18 persons). Extremes of age ie. less than 10 years and more than 90 years are rarely affected. The average age of presentation was 45.18 years.

80% of the patients affected were males. The male:female ratio was 4:1. This is comparable to the findings in the current literature.

2. CLINICAL SYMPTOMATOLOGY

Abdominal pain is the most common presenting symptom (91%) followed by vomiting (71%) and distention of abdomen (53%).

The most common sign was tenderness which was present in (89%) of the cases, followed by guarding & rigidity which was present in 72% of patients.

DISEASE SPECTRUM

Of the 100 cases the commonest cause of acute intestinal obstruction was obstructed hernia (74%) which was comparable to the literature. Adhesive obstruction was present in 11% of cases, and is the second commonest cause.

Among the Hernias, inguinal hernias were the most commonly implicated type accounting for 63 cases (63%). Other hernias account for comparatively lesser percentage.

The inguinal hernias are the most common type of hernia associated with strangulation of the bowel which occurred in 7 cases out of which inguinal hernias are found in 5 cases and incisional hernias in 2 cases.

Investigational aids

Plain X-Ray abdomen was taken for all the 100 cases of intestinal obstruction. The most common finding in the X-Ray was multiple fluid levels (>3) which was seen in 87 patients, dilatation of bowel loops proximal to intestinal obstruction was seen in 42 cases, 4 cases had coffee bean appearance. X-Rays were inconclusive in another 7 cases.

Ultrasound abdomen was preformed, it revealed dilated bowel loops in 42 cases, free fluid in 15 cases and in majority of the patients (42 cases) the results are inconclusive. The ultrasound had little role to play in the diagnosis of intestinal obstruction.

Surgical Procedure adopted

Reduction and hernia repair was the most common surgical procedure performed accounting for 61 cases (61%) followed by resection and anatomosis 14 cases (14%).

Adhesiolysis was done in 8 cases, colostomy in 5 cases (5%) and other procedures were performed in the remaining patients.

The surgical outcome

Majority (69%) of patients had an uneventful recovery. 3 patients died in the postoperative period.

LITERATURE REVIEW

1. Acute intestinal obstruction in adults in “Kumasi Ghana” ohene yeboah

Adippah. E, Gyasi, Sarpong K. Department of Surgery, Komfo Anokye Teaching Hospital, Kwame, Kumasi, Ghana.

They conducted a prospective study, between 1998-2003 of 652 cases of intestinal obstruction of which 412 (63.2%) were due to strangulated external hernias and 176 (27.2%) were due to post operative adhesions which is comparable to our study. (18)

2. Hernias are the most common cause of strangulation in patient with small bowel obstruction.

Thedioha.Y, Alani A, Modak P, Chong P,

O’Dwyer PJ, University of department of surgery western infirmary Dumbarton road, GLASGOW.

Hernias remain the most frequent cause of strangulation in patients presenting with intestinal obstruction (11). This study is comparable with our study.

CONCLUSION

The study of acute intestinal obstruction was aimed at identifying the etiological factors, mode of presentation, surgical procedures employed with relevant investigational procedures.

Acute intestinal obstruction is one of the most common emergencies requiring surgical intervention. A proper and complete bedside clinical examination by the surgeon is more superior than investigational procedures. Hernias continues to be the commonest cause for obstruction and strangulation. A delay in diagnosis could be fatal. Adhering to the basic principles of preoperative preparation like nasogastric suctioning, correcting dehydration and electrolyte imbalances can significantly lower the morbidity and mortality. Surgical intervention remains the cornerstone of treatment. With timely management, most patients have an excellent prognosis.

PROFORMA

Name: Age: Sex:

Inpatient No.: Ward:

I PRESENTING FEATURES

1. Abdominal pain
2. Vomiting
3. Obstipation
4. Abdominal distension

II. PAST HISTORY

H/O PREVIOUS SURGERY

H/O TUBERCULOSIS

III. MENSTRUAL AND OBSTETRIC HISTORY

IV. FAMILY HISTORY

H/O TUBERCULOSIS

H/O MALIGNANCY

V. GENERAL EXAMINATION

PULSE RATE - TACHYCARDIA

BLOOD PRESSURE – HYPOTENSION

SIGNS OF DEHYDRATION

ANAEMIA

FEVER

VI. EXAMINATION OF ABDOMEN

1. Surgical Scar
2. Visible intestinal peristalsis (or) Colonic peristalsis
3. Abdomen distension
4. Tenderness
5. Guarding
6. Rigidity
7. Mass Palpable
8. High pitched tinkling bowel sounds / silent abdomen
9. Hernial Orifices
10. PER RECTAL EXAMINATION – Roomy / empty rectum, blood, a mass or a blummer shelf nodule palpable
11. PER VAGINAL EXAMINATION – Evidence of gynaecological Malignancy (or) inflammatory lesion

VII. EXAMINATION OF OTHER SYSTEMS

VIII. INVESTIGATIONS

Hb%

Total count

Differential count

Erythrocyte sedimentation rate

Blood Urea

Blood sugar

Serum creatinine

Serum electrolytes

X-Ray abdomen erect

Chest X-Ray PA view

Ultrasonogram abdomen

IX. MANAGEMENT

1. Fluid and Electrolytes repletion
2. Decompression of gastro intestinal tract
3. Antibiotics
4. Timely surgical invervention

X. FOLLOW UP

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MASTER CHART

S.No.	Name	IP No.	Age	Sex	Clinical Presentation							Investigations		Diagnosis IOP findings	Procedure	Out come grade
					Pain	Abd. distension	Vomiting	Obstipation	G/R	T	Dehydration	AXR	USG			
1	Balan	12183	42	M	+	-	+	-		+	+	MFL	-	Rt. obst. ing. hernia	RHR	I
2	Mannappan	13458	30	M	+	+	+		+	+	+	DBL	DBL	Ladd's band	Band Release	II
3	Boopalan	14229	45	M	+	-	+	-	+	+	-	-	-	Rt Obst. Ing. hernia	RHR	I
4	Ramesh	17548	27	M	+	+	+		+	+	+	MFL	DBL	Adhesive obstruction	Adhesiolysis	II
5	Arumugam	15280	30	M	+	+	+	+	+	+	+	MFL DBL	DBL FF	Adhesive obstruction small bowel ischemic stricture	Adhesiolysis resection and anastomosis of small bowel	II
6	Vadivel	16/09	20	M	+	+	+	+	+	+	-	MFL DBL	DBL	Adhesive obstruction	Adhesiolysis	I
7	Ganesan	16037	40	M	+	+	-	-	-	+	-	-	-	Rt. Obst. Ing hernia	RHR	I

8	Govindasamy	17870	65	M	+	+	+	+	+	+	+	MFL DBL	DBL FF	SMA thrombosis Small bowel ischemic stricture	Resection and Anastomosis of small bowel	IV
9	Venkatesan	19783	33	M	-	+	+	+		+	-	MFL	-	Lt Obst. Ing. hernia	RHR	I
10	Ramalingam	26590	48	M	+		+			+		DBL	DBL	Rt. Obst. Ing hernia	RHR	I
11	Shankar	20514	25	M	+	+				+		MFL	-	Rt. Obst. Ing. hernia	RHR	I
12	Thomas	20588	70	M	+	+	+	+	+	+	+	MFL	DBL	Adhesive Obstruction	Adhesiolysis	I
13	Chandran	21210	60	M	+	-	-	-	-	+	-	DBL	-	Rt. obst. Ing. hernia	RHR	I
14	Rajamani	21341	42	M	+	-	+	-	+	+	-	-	-	Lt. Obst. Ing. hernia	RHR	I
15	Mohammed Kasim	21529	16	M	-	+	+	+	+	-	+	MFL DBL	DBL	Carcinoma ascending colon	Rt. Hemi colectomy	IV

16	Kamala	21573	62	F	+	+	+	+	+	+	+	MFL DBL	DBL FF	Obstructed Incisional hernia Small bowel gangrene	Resection and anastomosis of small bowel with anatomical repair	II
17	Babu	21678	25	M	-		+			+		MFL	-	Rt. Obst. Ing. hernia	RHR	I
18	Munuswamy	21736	75	M	+	+	+	+	+	-	+	MFL DBL	DBL FF	Adhesive obstruction Small bowel gangrene	Adhesiolysis Resection and anastomosis of small bowel	III
19	Mani	23566	57	M	+	+	+	-	+	+	+	MFL	-	Rt. obst. Ing. hernia	RHR	I
20	Murugan	24212	25	M	+	-	-	-	+	+	--	MFL	-	Rt. obst. Ing. hernia	RHR	I
21	Ganesan	24290	45	M	+	+	-	-	-	+	-	MFL	FF	Rt. obst. Ing. hernia	RHR	I
22	Sampath	24372	32	M	+	-	+	-	+	+	-	MFL		Rt. obst. Ing. hernia	RHR	I

23	Varadhan	24511	40	M	+	+	-	-	-	+	+	DBL	DBL	Rt. obst. Ing. hernia	RHR	I
24	Nagappan	25131	40	M	+	+	-	-	-	+	+	MFL	-	Rt. obst. Ing. hernia	RHR	II
25	Sundaram	25043	45	M	+	+	-	-	-	+	-	MFL	-	Rt. obst. Ing. hernia	RHR	I
26	Manohar	25503	38	M	-		+		+	-	-	DBL	DBL	Lt. obst. Ing. hernia	RHR	I
27	Anitha	27078	17	F	+	+	+	+	+	+	+	MFL DBL	DBL FF	Splenic flexure growth with large bowel obstruction	Diversion colostomy	II
28	Govindan	27718	70	M	+	+	+	-	+	+	+	MFL DBL	DBL	Adhesive obstruction	Adhesiolysis	II
29	Periyasami	31503	20	M	+	+	-	-	-	+	-	DBL	DBL	Rt. obst. Ing. hernia	RHR	I
30	Seenu	31280	40	M	_	-	+	-		-	-	MFL	-	Rt. obst. Ing. hernia	RHR	I

31	Mani	32420	40	M	+	+	+	-	+	+	+	MFL DBL	FF	Small bowel volvulus and gangrene	Resection of Ileum and ileostomy	III
32	Ayee Gounder	33908	70	M	+	-	+	-	+	+	-	MFL	-	Lt. obst. Ing. hernia	RHR	I
33	Venkatesan	32824	35	M	+	-	-	-	-	+	-	MFL	-	Rt. obst. Ing. hernia	RHR	I
34	Ragavan	1031	53	M	+	+	+	-	+	+	+	MFL DBL	DBL	Adhesive obstruction	Adhesiolysis	II
35	Murugan	1262	40	M	+	-	+	-	-	+	-	-	-	Rt. obst. Ing. hernia	RHR	I
36	Srinivasan	860	61	M	+	+	-	-	+	+	-	DBL MFL	DBL	Rt. obst. Ing. hernia	RHR	I
37	Gunasekar	3456	42	M	+	-	+	-	-	+	-	MFL		Rt. obst. Ing. hernia	RHR	I
38	Yacub	4689	35	M	-	+	-	-	+	-	-	MFL DBL	DBL	Lt. obst. Ing. hernia	RHR	I
39	Gunasekaran	4962	24	M	+	-	-	-	+	+	-	DBL	DBL	Rt. obst. Ing. hernia	RHR	I
40	Neelamegam	2204	58	M	+	-	+	-	-	+	-	MFL	-	Lt. obst. Ing. hernia	RHR	I

41	Senthil Kumar	31073	21	M	+	+	+	+	+	+	+	CBA		Sigmoid volvulus	Colostomy	II
42	Mohana	8881	55	F	+	+	+		+	+	+	MFL DBL	DBL	Obstructed para umbilical hernia	Obstruction Release and anatomical repair	I
43	Perumal	8715	28	M	+	-	-	-	-	+	-	MFL	-	Lt. obst. Ing. hernia	RHR	I
44	Patchiammal	8184	67	F	+	+	+	+	+	+	+	CBA	DBL FF	Recurrent Sigmoid volvulus	Sigmoidectomy	II
45	Santhanam	8550	55	M	+	+	+		+	+	+	MFL DBL	DBL FF	Strangulated Rt. Ing.hernia	Resection and anastomosis of small bowel and herniorraphy	I
46	Sekar	36349	40	M	-	+	+		+	-		MFL	_	Rt. Ing. hernia	RHR	I
47	Karuppaiya	38289	45	M	+	-	-	-	-	+	-	MFL	FF	Rt. Ing. hernia	RHR	II
48	Anandan	38857	23	M	+	-	+	-	+	+	-	-	-	Rt. Obst. Ing. hernia	RHR	II

49	Nagaraj	40926	81	M	+	+	+	+	+	+	+	DBL MFL	DBL	Strangulated Lt. Ing. hernia	Emerg. Laparotomy with resection and anastomasis of small bowel and herniorraphy	III
50	Balaji	41176	21	M	+	+	+	+	+	+	+	CBA	DBL	Sigmoid Volvulus	Sigmoidectomy	I
51	Maharunisa	41212	29	F	+	+	+	-	+	+	-	MFL DBL	DBL	Rt. Obst. femoral hernia	RHR	I
52	Babu	994	27	M	+	-	+	-	+	+	-	MFL	DBL	Rt. Obst. femoral hernia	RHR	I
53	Aandal	940	70	F	+	+	+	-	+	+	+	DBL MFL	DBL	Obst. umbilical hernia	Mayo's repair	I
54	Jayalakshmi	1065	60	F	+	+	+	-	+	+	-	MFL	-	Obst. paraumbilical hernia	Anatomical repair	I

55	Kumar	1190	35	M	+	+	+	+	+	+	+	MFL DBL	DBL FF	Rt. side strangulated ing. hernia gangrene of small bowel	Emergency laparotomy with small bowel resection and anastomosis	II
56	Sivakumar	1504	32	M	+	-	+	-	-	+	-	MFL	-	Rt. Obst. Ing. hernia	RHR	I
57	Ramaniah	2323	65	M	+	-	-	-	-	+	-	MFL	-	Rt. Obst. Ing. hernia	RHR	I
58	Subramani	2911	71	M	+	-	+	-	-	+	-	MFL	-	Rt. Obst. Ing. hernia	RHR	I
59	Raja	3270	80	M	+	-	-	-	-	+	-	MFL	-	Rt. Obst. Ing. hernia	RHR	I
60	Mani	2638	52	M	-	+	-	-	-	-	-	MFL	-	Rt. Obst. Ing. hernia	RHR	I
61	Amirthalingam	35191	82	M	+	-	+	-	+	+	-	MFL	-	Lt. Obst. Ing. hernia	RHR	I

62	Malliga	17446	42	F	+	+	+		+	+	+	MFL DBL	DBL	Adhesive Int. Obstruction Ileal Stricture	Adhesiolysis Ileal resection and anastomosis	II
63	Shankar	14343	58	M	+	-	+	-	-	+	-	MFL	-	Lt. Obst. Ing. hernia	RHR	I
64	Poongavanam	40611	32	M	+	-	-	-	-	+	-	MFL	-	Lt. Obst. Ing. hernia	RHR	I
65	Shanti	14652	54	F	+	+	+	-	+	+	-	MFL	-	Rt. Obst. femoral hernia	Lockwood procedure	I
66	Williams	16337	32	M	+	-	-	-	+	+	-	MFL	DBL	Rt. Obst. Ing. hernia	RHR	I
67	Elumalai	18062	55	M	+	+	+		+	+	+	MFL	DBL	Adhesive obstruction Ileal stricture	Adhesiolysis Ileal resection and anastomosis	II
68	Ranganathan	18173	60	M	+	-	-	-	-	+	-	MFL	-	Rt. Obst. Ing. hernia	RHR	I
69	Suguna	19803	30	F	+	+	+		+	+	+	MFL DBL	DBL	Adhesive intestinal obstruction	Adhesiolysis	I
70	Balasundaram	20558	44	M	+	+	+	+	+	+	+	MFL	DBL	Ileocaecal TB	Rt.	II

												DBL		stricture	hemicolectomy	
71	Shanthi	21196	34	F	+	+	+	-	+	+	-	MFL DBL	DBL	Irreducible incisional hernia	Anatomical Repair	II
72	Palani	21444	20	M	+	+	+	+	+	+	+	MFL DBL	DBL	Small bowel volvulus	Resection and anastomosis	II
73	Ravi	21831	42	M	+	-	+	-	+	+	-	MFL	-	Rt. Obst. Ing. hernia	RHR	I
74	Pandiyan	22818	71	M	+	-	-	-	-	+	-	MFL	-	Rt. Obst. Ing. hernia	RHR	I
75	Kasthuri	23095	58	F	+	+	+	-	+	+	-	MFL	DBL	Obstructed paraumbilical hernia	Anatomical Repair	I
76	Prasad	23758	40	M	+	+	+	+	+	+	+	CBA	DBL	Sigmoid volvulus	Sigmoidectomy	III
77	Narayanasamy	24833	81	M	+	-	-	+	-	+	-	MFL	-	Lt. Obst. Ing. hernia	RHR	I
78	Ethiraj	26720	62	M	+	-	-	+	-	+	-	MFL	-	Lt. Obst. Ing. hernia	RHR	I

79	Lakshmi	27483	60	F	+	+	+	-	+	+	+	MFL DBL	DBL	Rt. strangulated Ing. hernia-small bowel gangrene	Emergency laparotomy and resection and anastomosis of small bowel	II
80	Mari	27858	38	M	+	-	+	-	+	+	-	MFL	-	Lt. Obst. Ing. hernia	RHR	I
81	Seeman	31287	52	M	+	-	-	+	+	+	-	MFL	-	Rt. Obst. Ing. hernia	RHR	I
82	Gothandam	33174	35	M	+	-	+	-	-	+	-	MFL	-	Rt. Obst. Ing. hernia	RHR	I
83	Purushothaman	7043	72	M	+	-	-	-	+	+	-	MFL	-	Lt. Obst. Ing. hernia	RHR	I
84	Mala	9631	24	F	-	+	+	-	+	-	-	MFL DBL	DBL	Adhesive Obstruction	Adhesiolysis	I
85	Nagammal	8126	70	F	+	+	+	-	+	+	-	MFL	DBL	Obstructed paraumbilical hernia	Anatomical Repair	II
86	Kumaravel	9640	42	M	+	-	-	-	-	+	-	-	-	Rt. Obst. Ing. hernia	RHR	I

87	Sarala	9919	45	F	+	+	+	-	+	+	-	MFL DBL	DBL	Obstructed Incisional hernia	Anatomical Repair	I
88	Krishnammal	8341	60	F	+	+	+	+		+	+	MFL DBL	DBL	Splenic flexure growth	Transverse loop colostomy	I
89	Murugesan	10723	27	M	+	-	-	-	-	+	-	MFL	-	Rt. Obst. Ing. hernia	RHR	I
90	Subramani	11066	52	M	+	-	-	-	-	+	-	MFL	-	Rt. Obst. Ing. hernia	RHR	I
91	Vallithai	26881	29	F	+	+	+	+	+	+	+	MFL DBL	DBL FF	Rectal Carcinoma Large bowel obstruction	Laparotomy and sigmoid loop colostomy	IV
92	Jayammal	12479	76	F	+	+	+	+	+	+	+	MFL DBL	DBL FF	Rectal Carcinoma Large bowel obstruction	Transverse loop colostomy	II
93	Palani	11197	30	M	+	+	+	-	+	+	+	MFL DBL	DBL FF	Ileocaecal TB stricture	Rt. hemi colectomy	II

94	Mannu	11283	80	M	+	-	-	+	-	+	-	MFL	-	Rt. Obst. Ing. hernia	RHR	I
95	Dhandapani	12567	55	M	+	-	+	-	-	+	-	MFL	-	Rt. Obst. Ing. hernia	RHR	II
96	Krishnamoorthy	15195	40	M	+	-	-	-	+	+	-	-	-	Rt. Obst. Ing. hernia	RHR	I
97	Gopiraj	15229	48	F	+	+	+	+	+	+	-	MFL		Lt. Obst. Ing. hernia	RHR	I
98	Gnammal	1587	49	F	+	+	+	+	+	+	+	MFL DBL	DBL FF	Strangulated Incisional hernia gangrene of Ileum	Emergency laparotomy Ileal resection and anastomosis and Anatomical repair	III
99	Shanmugam	16005	67	M	+	-	+	-	+	+	-	MFL	-	Rt. Obst. Ing. hernia	RHR	I
100	Thirumalai	16201	21	M	+	-	+	+	+	+	-	MFL DBL	DBL	Lt. side obstructed Ing. hernia gangrene of jejunum	Resection anastomosis of jejunum and hernioplasty	III

MFL - Multiple Fluid Level

DBL - Dilated Bowel Loop

CBA - Coffee Bean Appearance

RHR - Reduction and hernia Repair

FF - Free Fluid

G/R - Guarding / Rigidity

T - Tenderness

AXR - X-Ray abdomen erect

USG - Ultrasonogram

IOP - Intra Operative